

Clostridial diseases of animals in New Zealand

In the past, clostridial diseases were widespread in New Zealand and one of the very early activities of Wallaceville Agriculture Research Centre was to develop and produce blackleg vaccine. Subsequently this vaccine formed the basis for Mr Lou Fitch to establish one of the first commercial vaccine laboratories in New Zealand, Trentham Veterinary Laboratory, later known as Tasman Vaccine Laboratories (TVL) and now operated by Pitman-Moore.

Nowadays, with the use of clostridial vaccines so widespread, these diseases have decreased as a cause of economic loss. However, most of the causative organisms are normal gut or soil inhabitants still present in the environment, lying in wait for the unwary. Most of the pathogenic clostridia are present in New Zealand.

The isolation of some clostridia from soil can be difficult, but Table 1 compares the result of a study in New Zealand with one in Libya. Table 2 shows the distribution of isolates in different soil environments in New Zealand. These tables have been adapted from Professor Don Bacon's MSc thesis.¹

Clostridium tetani

The organism causing tetanus, *Cl. tetani*, is widespread throughout New Zealand. It is commonly thought by lay people to be 'bad where horses have been kept'. However, human intestinal carriage rate of *Cl. tetani* is at least as high as that of horses!² The organism is most common in cultivated land, less so in grazing land and cattle yards, and least in virgin soil. The lack of association between tetanus and domestic animals is reinforced by the fact that tetanus is common in Papua New Guinea where, until recently, there were no cattle, sheep or horses.²

Clostridium botulinum

Six main types of this organism, the cause of botulism, are known. These are known as types A to E. Types A and B are important causes of food-associated, wound and infant human botulism overseas and also cattle botulism in Australia. They are not known to cause animal disease in New Zealand. However, there has been one occurrence of type A botulism in humans here (R Alexander, New Zealand Communicable Disease Centre, personal communication).

Cl. botulinum type E tends to be associated with lake and river sediments in United States and Canada, with human type botulism usually resulting from imperfectly canned fish. It has not been recorded in New Zealand. Type C botulism has been confirmed in animals and birds in New Zealand. Until recently, its occurrence was thought to be confined to mortalities in water fowl associated with falling water levels and warm sum-

mer/autumn temperatures.³ However, recently there have been a number of cases of clinically typical botulism in dogs in the Northern half of the North Island.⁴ Some of these have been secondary to eating dead carcasses, but at least one was said to have occurred after the dog swam in a muddy pond.

It appears, therefore, that *Cl. botulinum* type C botulism could be an emerging disease in domestic animals in New Zealand.

It could be possible for other types to become established in this country, perhaps in sewage areas (where, incidentally, some of the first wild fowl type C outbreaks occurred).³ Such introduction could result from intestinal carriage in humans who acquired the organism overseas.

Cl. difficile

This is a recently-recognised pathogen which causes syndromes such as diarrhoea and dysentery in children. It has been reported as a cause of illness in foals.⁵ The organism is known to cause disease in humans and appears regularly in the monthly reports of the New Zealand Communicable Disease Centre. Ruakura

Animal Health Laboratory obtained a positive *Cl. difficile* toxin test result in a sample of foal faeces. Distribution of the organism is uncertain, but it is believed to primarily live as an intestinal commensal (see also *Cl. perfringens* type D), so is probably widespread. Its significance as a cause of animal disease in New Zealand is unknown, mainly because it has been tested for so seldom.

The gas gangrene group

Cl. chauvoei (*feseri*), *Cl. septicum*, *Cl. perfringens* (*welchii*), *Cl. oedematiens* (*novyi*) are all widespread in nature. They are commonly found in the gut, and ingested spores may be absorbed and remain latent in body tissues. Post-mortem invasion and/or spore activation in response to falling redox potential of tissues after death lead to rapid multiplication in carcasses.

A British study showed that *Cl. oedematiens* could be demonstrated in 75%, and *Cl. septicum* in 17%, of livers from abattoir slaughtered sheep.⁶ A similar study in New Zealand could not reproduce these isolation rates, but the procedures used were different.⁷

A small study carried out in New

Table 1: Comparison of *Clostridia* isolated from New Zealand soils with those of cultivated areas in Libya

Organism	Libya* (%) n = 26	New Zealand** (%) n = 24
<i>Cl. perfringens</i>	22	21
<i>Cl. oedematiens</i>	8	6
<i>Cl. septicum</i>	4	1
<i>Cl. tetani</i>	6	1

* Specimens obtained from towns and villages in Libya.
** Specimens from domestic gardens, roadside verges, pastures, orchards, bush in New Zealand.

Table 2: Frequency with which species of *Clostridia* were isolated from the different soil types

Soils	n	<i>Cl. perfringens</i>	<i>Cl. oedematiens</i>	<i>Cl. tetani</i>	<i>Cl. septicum</i>
Domestic garden	7	6	1	-	-
Roadside	7	5	3	-	-
Pasture	5	5	2	1	-
Orchard	2	2	-	-	-
Swamp	2	1	1	-	1
Alpine	4	1	-	-	-
Non-fertile	4	1	-	1	-
Bush	3	3	-	-	-
TOTAL	34	24	7	2	1

Zealand by Margery Carter (unpublished) demonstrated *Cl. septicum* and *Cl. oedematiens* in aseptically collected ribs of three of eight euthanatised sheep. In other studies she showed that tissue samples (liver, muscle and rib) collected from euthanatised sheep, contained *Cl. perfringens*, *Cl. septicum* and/or *Cl. oedematiens* 24 hours after death.

These findings confirm that, in the absence of typical lesions, isolation of members of the gas gangrene group of clostridia provides no meaningful information for investigation of a disease problem.

Clostridium chauvoei

The bacterium causing the condition blackleg, *Cl. chauvoei*, is present throughout New Zealand. Disease is more common in some localities. Some farms are considered to be 'bad for blackleg'. Lesions at unusual sites can be missed, particularly myocardial blackleg in cattle and tongue/oral lesions associated with teething/trauma. Blackleg can occur at vaccination sites, possibly because previously ingested spores latent in muscle are activated by lowered redox potential caused by the trauma of injection.

Clostridium septicum

The cause of malignant oedema, *Cl. septicum* is widespread throughout

New Zealand. Disease is seen mainly as navel infection in lambs and parturient 'blood poisoning' in ewes. Clinical malignant oedema is seldom seen and difficult to prove when suspected. Braxy, due to *Cl. septicum* spore activation in the abomasal wall after eating frozen root crops, has not been described in New Zealand.

Clostridium oedematiens

There are four main types (A-D) of *Cl. oedematiens*. Fluorescent antibody procedures are generally used in Animal Health Laboratories to detect *Cl. oedematiens* and this does not differentiate types.

Type A is one of the classical gas gangrene wound organisms of humans. It has not been isolated from New Zealand soils.¹ It is not generally regarded as a significant animal pathogen but is a rapid post-mortem invader.

Type B *Cl. oedematiens* causes black disease (mainly in sheep). Spores are ingested and remain quiescent in liver until activated by tissue damage, classically larval fluke migration. Black disease is known to have occurred in this country and the organism is widespread in New Zealand soils.¹ With the widening distribution and, possibly, also increased severity of liver fluke infestations, the disease may become more common (depending on vaccination regimens). It is also a rapid post-mortem invader.

'Big head', seen in ram lambs in Aus-

tralia, has not been described in New Zealand.

Type C is essentially non-toxigenic and is not known to cause animal disease. Occurrence in this country is unknown.

Type D *Cl. oedematiens* is also known as *Cl. haemolyticum* and causes bacillary haemoglobinuria in cattle. It has been recorded here^{8,9} but is uncommon. It could become more common if liver fluke infestations in cattle increase, although the association between bacillary haemoglobinuria and liver fluke is not as clearly established as with black disease.

Clostridium perfringens

There are again five main types of *Cl. perfringens*, labelled A through E. Only types A and D are known to occur here. The epidemiology of disease differs between types. The typing is based on the presence or absence of four main toxins (alpha), (beta), (epsilon) and (iota). They all have varying amounts of toxin.

Type A has alpha toxin alone

Type C also has beta toxin

Type D also has epsilon toxin

Type E also has iota toxin

Type B also has both beta and epsilon toxin

Enterotoxaemia (pulpy kidney) of ruminants

Caused by *Cl. perfringens* type D, pulpy kidney disease occurs throughout New Zealand. The organism is mainly a gut inhabitant. It does not thrive in the soil environment, where type A predominates. Type D does not persist in soil beyond 12 months after livestock have been removed.¹⁰

The epsilon toxin is activated by trypsin in the gut. It must be present at high concentration for a reasonable period before gut permeability is damaged sufficiently for absorption to occur. Circulating antitoxin, actively or passively acquired, is highly effective in neutralising it. Therefore the presence of epsilon toxin in gut contents is not, in itself, sufficient to diagnose enterotoxaemia.

Cl. perfringens types B and C are not known to occur in New Zealand and there is no evidence that the clinical syndromes of lamb dysentery (type B) or struck (type C) occur here. There was an occasion many years ago when a New Zealand medical institution reported isolation of type B, but this was never proven conclusively.

Because the beta toxin is inactivated by trypsin it is difficult to demonstrate in gut contents. For this reason bacterial isolation and strain typing are essential in demonstrating that disease is due to beta toxin-producing types. Such procedures are not carried out regularly at New Zealand Animal Health Laboratories, but would be undertaken if there was clinical evidence to suggest lamb dysentery or struck. Types B and C are common in sheep, cattle and goats in

Europe² and both type C and type D enterotoxaemia can cause problems in camelids in North and South America.¹¹ Since types B and C also are normal intestinal inhabitants, it is quite possible that they will be, or have been, introduced into New Zealand with any of the above species. Type C could have public health significance. It has been implicated as the cause of enteritis necroticans in Germany and New Guinea.

Type E has been recovered from sheep and cattle but its pathogenicity has not been clearly established. It is not known to occur in New Zealand.

Cl. perfringens type A is the other major gas gangrene organism of humans. Heat resistant forms are also responsible for outbreaks of food poisoning. It persists in soil and is also found commonly in the gut of animals. Both heat resistant and normal strains have a world wide distribution, including New Zealand. In animals its role as a pathogen is rather difficult to assess. Because of its widespread distribution, it has not been possible to establish cause and effect relationships clearly. It is considered to be the cause of occasional wound infection and haemorrhagic enteropathy of dogs fed spoiled food.¹² Both conditions occur in New Zealand. It may perhaps cause an enterotoxaemia/enteropathy syndrome in calves and, possibly, camelids (along with types C and D) but the literature on this condition is sparse.^{2,11}

There is experimental evidence that strains of *Cl. perfringens* type A differ in their pathogenicity. However, as with most other clostridial diseases, the interaction of host, environment and organisms is more important in the causation of disease than the presence of a particular strain of type A.

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